quently accompanied by pulmonary tuberculosis, although this latter coincidence seems to be without clinical significance except in diagnosis.

4. Cervical rib may occur without symptoms, or with symptoms so slight as not to constitute any inconvenience or disability; and this may be true even when the cervical rib is of unusual size or prominence, as in several of my series.

5. Symptoms may develop at any age, and with or without a history of injury, occupational strain, or other known etiological factor, but they are much more apt to develop later in life and without definite history of causation.

6. In certain cases of cervical rib the abnormal course of the subclavian artery may simulate aneurysm, but true aneurysm is probably infrequent in these cases, and can only be demonstrated by operation or at autopsy.

7. In those cases presenting symptoms of sufficient severity to warrant operation, the resection of the supernumerary rib under recent technique offers every reasonable prospect of complete recovery.

8. In some cases of cervical rib the only clinical significance which can be attached to the condition lies in the fact that the recognition of early pulmonary tuberculosis may be rendered difficult through the presence of the supernumerary rib obscuring definite signs at the apex underlying it; or, of equal importance, that the presence of cervical rib in some cases of suspected tuberculosis may lead to a mistaken diagnosis of apical lesion because of the confusing signs above the clavicle due to the presence of this anomaly.

THE POLYNEURITIC FORM OF ACUTE POLIOMYELITIS: A CLINICAL AND PATHOLOGIC STUDY.

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The question of the existence of a true polyneuritic form of acute poliomyelitis has never been satisfactorily determined. As far as I am aware no case has ever come to necropsy. Wickman in his article on "Acute Poliomyelitis" in Lewandowsky's *Handbook,* published in 1911, states "that he was unable to find in the literature a report of this type in which the peripheral nerves had been studied." Recently, I had the opportunity of studying a fatal case under the care of Dr. Pitfield.

A. S., female, aged twenty years, was admitted to the hospital on August 17, 1911. Nothing of importance was noted in the family or medical history. Her illness began several days previous to admission with nausea, vomiting, and fever, accompanied by severe pain in the back of the head and neck. A few days later pains and paralysis occurred in both lower limbs, especially the left. Physical examination of the heart and lungs showed no changes. The abdomen was somewhat distended.

The left lower limb was completely and the right lower limb was partially paralyzed, and only the foot could be moved. Pain on pressure over the nerve trunks was noted in both limbs. The left knee-joint was swollen. There was paralysis of the bladder.

No changes were found in the urine. The blood examination showed: Red blood cells, 4,600,000; white blood cells, 7200; hemoglobin, 80. The Wassermann reaction was negative.

August 29. General condition as on admission, save that the patient voids urine voluntarily.

September 18. Pupils equal; reacted to light and accommodation. There were no ocular palsies. The eye-grounds showed no changes. There was no involvement of the cranial nerves. She could move either arm freely, and in all directions. The biceps-jerk was normal. There were no sensory disturbances in the arms or hands. She could move the right foot and leg slightly. The left limb could not be moved at all. Both patellar tendon reflexes were absent. The knee-joints were swollen. Tactile sensation was diminished in both lower limbs. A pin-point was felt over the entire left lower extremity, but not so distinctly over the right lower limb above the knee. There was marked tenderness over the nerve trunks in the lower limbs.

September 24. The urine showed casts and albumin. The condition of the patient remained the same, except that the temperature rose on the thirty-third day, and remained between 102° and 104° until death.

History. A young adult female, without any previous illness, was suddenly seized with vomiting, fever, pain in the head and back, followed in several days by pain and paralysis in the lower limbs, together with paralysis of the bladder. The important physical symptoms were loss of patellar tendon reflexes and unsymmetric paralysis of both lower limbs, with partial areas of hypesthesia. There was bladder paralysis. In addition there was marked tenderness on pressure over the nerve trunks in the paralyzed extremities, which persisted until death, two months after the onset.

At necropsy the heart, lungs, spleen, and liver showed no changes. The kidneys showed cloudy swelling. A few punctate hemorrhages were seen in the visceral pericardium.

No exudate was noted in the meninges of the brain or spinal cord. The vessels were injected. The cord in the lumbar region was somewhat softer than in the other portions, and on section showed a reddish-gray area in both anterior horns.

Microscopic sections were made from various levels of the spinal cord and medulla oblongata. These sections were stained with thionin hemalum and acid fuchsin, by Weigert's stain, and by the Marchi stain.

Only one peripheral nerve and portion of one muscle were obtained and these were from the anterior aspect of the right thigh, near Poupart's ligament. A portion of the nerve was placed immediately in osmic acid and teased after twenty-four hours. Another portion was placed in Müller's fluid and studied by the Weigert, hemalum, and acid fuchsin stains. There was no evidence of an acute or subacute inflammatory process, and no distinct degenerative changes were noted. The hemalum-acid-fuchsin stain showed slight increase in the connective tissue. There was no round-celled infiltration. The osmic acid showed slight swelling of the fibers, some granulation of the myelin, but no breaking up into droplets. Most of the fibers were normal with Weigert's stain.

The section of muscle showed no round-celled infiltration, no swelling or atrophy of fibers. The striations could be seen, but were not sharply defined.

In the medulla oblongata no changes were noted.

The cervical and thoracic areas of the spinal cord showed no changes.

In the lumbar area stained with the hemalum-acid-fuchsin the meninges showed an intense round-celled infiltration.

The pial vessels were dilated and intensely engorged with blood. Around and within the anterior and posterior roots the round-celled infiltration was intense, and the capillaries were intensely dilated.

The anterior horns showed the characteristic picture of the subacute stage of an acute poliomyelitis.

The left anterior horn was more involved than the right. The capillaries were injected, the round-celled infiltration was marked, and the cells showing a clear protoplasmic area around the nucleus.

Many lymphoid cells, a few connective-tissue and polymorphonuclear cells were also seen. The ganglion cells had practically disappeared as only a few were seen, and those remaining were atrophied. With the Nissl stain the disappearance of the ganglion cells and the varying grades of degeneration of those remaining were most marked.

No changes were seen in the posterior horns or in the white matter. The Weigert stain showed no sclerosis.

These changes were noted in the second, third, fourth, and fifth lumbar segments.

Pathologic History. The changes found in the lumbar region of the spinal cord were those of an acute poliomyelitis in the reparative stage.

The anterior horns were practically destroyed. Intense inflammation was noted in the meninges and around the posterior roots.

Most interesting was the exudate and inflammation of the spinal roots.

The peripheral nerve showed no evidence of a primary neuritis, and the changes present were probably secondary in character.

The diagnosis of this case was difficult. The possibility of a true multiple neuritis had to be excluded. Pain on pressure over the nerve trunks, which is emphasized so much in text-books, could not be considered as a differential symptom.

True multiple neuritis develops slowly, fever is usually absent, special groups of muscles are affected, and the distribution is more symmetric. The absence of sore throat and any history of diphtheria precluded a diphtheritis neuritis, the paralysis in this also occurs much later than the throat disorder; a neuritis of other type, could not with certainty be excluded. The diagnosis of the polyneuritic form of acute poliomyelitis was substantiated by the presence of several other cases in the same vicinity, the unsymmetric involvement of the paralysis, and the absence of cranial nerve involvement, though there are cases of multiple neuritis in which only the spinal nerves are affected, and cases of acute poliomyelitis have been reported with cranial nerve paralysis.

The resemblance of multiple neuritis to poliomyelitis has been known ever since Leyden recognized a clinical relationship.

This similarity in the clinical pictures of the two diseases was recognized in the chronic form of the spinal disease, but recent studies have established the resemblance to acute poliomyelitis.

Cases were described clinically by Strümpell and Barthelmes in 1900. Wickman in 1905 reported a few cases, and in the epidemics observed by Hartman, Foerster, Müller, Mills, and Spiller this form has been recognized. In Foerster's cases everyone showed pain and tenderness on pressure over the nerve trunks, which continued for months. At least clinically there seems to be no question of a polyneuritic form. These cases are remarkable in the prominent part played by the pain over the nerve trunk. After a shorter or longer interval, varying from days to several months, this pain disappears. These cases may recover completely or show permanent paralysis; in rare instances they are fatal.

Those cases in which the pain resolves itself into a hyperesthesia of the skin or on movement of the joints or limbs should not be included in the polyneuritic form. Many cases of acute poliomyelitis may show this at the onset of the disease.

In some of the polyneuritic forms, objective sensory symptoms may be present, such as hypesthesia and disturbances of pain and temperature.

Pathologic evidence of this form is wanting. The peripheral nerves have never been examined in any such case. Cases of acute and subacute poliomyelitis not of the neuritic type have been reported in which the peripheral nerves were studied. In the cases of Redlich, Monkberg, Mott, and others the nerves were found degenerated. This degeneration could be attributed to a secondary change, owing to the long duration of the cases (from thirteen days to several months).

Harbitz and Schell's cases of acute poliomyelitis, with pain, do not answer to the polyneuritic type, though their findings are of interest.

In their Case VI, a boy, aged six years, had headache, pain in the neck, and pain in the arms. Examination of the brachial plexus and the vagus nerve showed no inflammation or degeneration.

In their Case VIII, one of poliomyelitis, with bulbar symptoms, in which pain played a prominent part at the onset, the vagus, phrenic, and cervical plexus showed no changes.

Their Case X showed an acute onset, with headache, fever, pain in the limbs, and bladder paralysis. No changes were noted in the peripheral nerves.

Wickman believes that the peripheral nerves are never involved and that the pain is due to the meninges, while Raymond held the view that the whole peripheral neuron is affected.

Strümpell, while admitting the involvement of the peripheral neuron, believes that the two diseases are distinct, in that in the spinal type we are dealing with a focal lesion, while in the neuritic form we have the effect of a toxic disturbance in the general circulation.

The pathologic evidence was wanting of any primary degeneration of the peripheral nerve in my case. There were no inflammatory changes. That only one nerve was studied renders this evidence insufficient, but it is reasonable to assume that even this should have shown some neuritis.

Changes may, however, be absent even in true neuritis. Spiller has examined the nerves in different cases of peripheral neuritis, and in some instances he has failed to find any change. This he attributes to the unequal distribution of the inflammatory process, though clinically in his cases the pain was felt along the entire nerve. The more central portions of a nerve may escape when the peripheral portions are diseased. Swelling of the nerve at one portion would produce pain throughout, yet the nerve above the swelling might not show any changes.

Pathologic evidence was not lacking to explain the pain in the case reported. The intense inflammation in the meninges, quite marked around the posterior roots, and the swelling of these roots, on account of the exudation of the round cells within them, as well as the intense engorgement of their vessels, might well account for the pain. The tenderness along the nerve trunks could easily result from this central irritation. In this connection it would be interesting to know whether in other diseases of the spinal cord such as tumor, etc., in which the spinal roots are irritated, tenderness could be elicited along the nerve trunks. That it occurs usually in acute meningitis is well known.